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Research Article

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Refractory Lesional Parietal Lobe Epilepsy: Clinical, Electroencephalographic and Neurodiagnostic Findings

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ABSTRACT

Introduction: Specialized centers, in the management and surgical treatment of medically refractory epilepsy, emphasize the importance of differentiating the varieties of localization related epilepsies. There has been considerable recent interest in temporal and frontal lobe epileptic syndromes and less attention has been paid to parietal and occipital lobe epilepsies.

Methods: Here we report the clinical, electroencephalographic and neuroimaging characteristics of 46 patients with medically refractory lesional parietal lobe epilepsy who have been followed up for 1-10 years.

Results: In this study auras were reported in 78.3% of the patients and included sensory symptoms (72.2%), headache (36.1%), nausea and vomiting (36.1%), psychic symptoms (36.1%) and visual symptoms (16.6%). The most common ictal behavioral changes were paresthesia (69.6%) and focal clonic activity (39.1%). Tonic posture, various automatisms, head deviation, staring, sensation of pain and speech disturbances occurred to a lesser extent. Simple partial seizures were present in 69.6%. Complex partial seizures occurred in 43.5% and secondary generalized tonic clonic seizures were reported in 58.7% of the patients. Interictal routine EEG disclosed abnormal background activity in 1/3 of the patients. Nonlocalising epileptiform abnormalities were found in 34.8% of the patients. EEG findings were normal in 34.8% of the patients. The most common presumed etiologic factors were as follows: posttraumatic encephalomalacia, stroke, tumor, malformation of cortical development, atrophy, and arteriovenous malformation.

Conclusion: Clinical, electrophysiological and neuroimaging features of the lesional symptomatic partial epilepsy patients may help us to localize the seizure focus in some patients with cryptogenic partial epilepsy. So that, the timing decision of the parietal lobe sampling with more invasive techniques like intracranial electrodes prior to epilepsy surgery would be easier.

Keywords: Parietal lobe, epilepsy, aura, EEG, MRI

INTRODUCTION

Epilepsy centers, specialized in the management and surgical treatment of medically refractory epilepsy, emphasize the importance of differentiating the varieties of localization related epilepsies (1). The traditional attempts to sub-classify these epilepsies have focused on their lobe of origin (2). The requirements for accurate localization include patients' medical history and neurological examination, EEG investigations, clinical seizure evaluation, neuropsychological studies and detailed neuroimaging (3). There has been considerable recent interest in temporal and frontal lobe epileptic syndromes and less attention has been paid to parietal and occipital lobe epilepsies (4). Knowledge about the clinical and electrical manifestations of parietal lobe seizures is based mainly on case studies and occasional larger series (2,3,5,6,7,8,9).

The incidence of parietal lobe seizures has been reported as almost 6% of all partial seizures (10). Epileptic seizures of parietal lobe origin are heterogeneous and mainly characterized by the presenting auras. Like all partial seizures, parietal lobe seizures consist of subjective and objective components. The most common subjective sensations or auras of parietal lobe seizures are paresthesia, usually numbness and tingling, but also a sensation of "pins and needles" and rarely crawling or itching (2).

When symptoms such as paresthesia or pain occur prominently and early in partial seizures, the origin of parietal lobe should be suspected. However, most patients with parietal lobe seizures have no symptoms or signs suggesting the parietal lobe. These patients can present with misleading findings because of the absence of detectable epileptogenic lesions. This results in erroneous localization, which can in turn lead to ineffective surgical intervention (2). Additionally, parietal lobes are large, diffuse structures, so the potential for sampling error is high. Even when parietal lobe seizure origin is suspected in the absence of a structural lesion, documenting this with invasive EEG monitoring can be difficult (2).

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Table 1. Characteristics of 46 lesional symptomatic parietal lobe epilepsy patients

No	Sex/ age on admission	Onset (years)	Medical History	Seizure type	Surface EEG	CT and /or MRI	Diagnosis	Treatment (surgery, RT, ChT, AED)	Prognosis
1	Fe, 35	35	A: right arm-leg numbness, dysarthria, right hand weakness S: right peroral focal onset, unconsciousness	SGTCS	1. L TP background abnormality 2. L TP slow and spike wave activity and PLED	L P 3 cm diameter cystic mass lesion	Tumor Pathology Low grade glial tumor	Tumor resection ChT+RT+PT	NA
2	Fe, 60	60	A: - GTCS	GTCS	1.R CTP slow wave 2.R CT paroxysmal anomaly	R P menin-gioma	Tumor Pathology Meningioma	Tumor resection+ PT	Seizure free under AED
3	Fe, 38	38	A: left facial pain, light-headedness, headache, left tinnitus, right arm and leg pain. S: right peroral focal con-vulsion, unconsciousness	CPS SGTCS	Normal	L P mass lesion	Tumor Grade 1 astrocytoma	Tumor resection ChT+RT+PT	NA
4	Fe, 20	20	A: - L focal motor seizure SGTCS during sleep	SPS SGTCS	1.R posterior background anomaly, paroxysmal activity 2. R posterior paroxysmal activity	R P low grade glial tumor	Tumor Pathology Low grade astrocytoma	Tumor resection ChT+ RT+PT	NA
5	M, 33	33	A: - Convulsion of right arm and contraction of right peroral region	SPS SGTCS	Paroxysmal anomaly at vertex and T regions	L P astrocytoma	Tumor Pathology astrocytoma	Tumor resection RT+PT	NA
6	M, 41	41	A: - SGTCS	SGTCS	Bilateral T slowing of background rhythms, left paroxysmal anomaly	L parietal tumor	Tumor Pathology astrocytoma	Tumor resection RT+ PT	NA
7	M, 36	35	A: - SGTCS	SGTCS	N	L P noduler lesions	Nodular lesion	Close follow-up+MT	NA
8	M, 19	14	Head injury, A: left arm , numbness feeling crash S: SGTCS	SPS SGTCS	N (4 times)	R P focal gliotic lesion encephaloma-lacia	Post traumatic encephaloma-lacia	MT	SGTCS <1/year
9	Fe, 18	12	Head injury, 5 monthsold Aura: Right arm dullness. S: clonic convulsion at right arm and face, vocalisation	SPS CPS	N	L P encephalo-malacia	Post traumatic encephalo-malacia	PT	SPS 4 / month
10	M, 19	15	18 months old head injury A: epigastric raising, thougths of being a scary place S: tonic neck convulsion on awake and sleep.	SPS CPS SGTCS	R FT epileptiform anomaly with nasopharyngeal electrodes	R P posttraumatic lesions at vertex level	Post traumatic encephalo-malacia	MT	Aura 1-2/ month
11	M, 44	43	Head trauma at 43yo A: Headache like wearing a helmet. S: lag, oral-hand automatism left peroral contraction, aphasia	CPS	N	R P cortical hyperintense lesion	Post traumatic encephalo-malacia	MT	Seizure free with AED
12	M, 19	12	Dystosia, mental retardation, head injury at 18 months. A:Numbness of left arm-leg S: Left facial twitching, bilateral tonic convulsion	SPS SGTCS	L>R bilateral CT paroxysmal epileptiform anomaly	L P cystic atrophic lesion	Post traumatic encephalo-malacia	PT	Aura 1-2/ month SGTCS 1/3-4 years

Table 1. Characteristics of 46 lesional symptomatic parietal lobe epilepsy patients (continued)

No	Sex/ age on admission	Onset	Medical History	Seizure type	Surface EEG	CT and /or MRI	Diagnosis	Treatment (surgery, RT, ChT, AED)	Prognosis
13	Fe, 34	34	Head injury and intracerebral hemorrhage at 18 months old. A: Tingling of left arm, flashing	SPS, SGTCS	1.Bilateral slowing of background rhythms, R T focal epileptiform anomaly 2.R T PLED	R Post P encephalo-malacia	Post traumatic encephalo-malacia	MT	Aura 4-5/ month, GTCS 1/year
14	M, 21	3	Dystosia, perinatal injury A: numbness of right hand, nausea, oral automatism. Right focal seizure at 3 yo, 3 status epilepticus, Shunt operation at 16 yo	SPS CPS SGTCS	1,2, 4 Slowing of background rhythyms, L CT, L PO epileptiform anomaly 3. L generalised epileptiform anomaly.	Hydrocephaly, L P porencephalic cyst, atrophy	Post traumatic encephalo-malacia	MT	NA
15	Fe, 18	10	Intracranial cyst operation at 13 yo, A: Numbness of left arm. S: Left arm convulsion then SGTCS	SPS SGTCS	1.R CT paroxysmal anomaly 2.N 3.R CT slowing of background rhythms	R P hyperintense lesions related to operation	Sequelaencepha-lomalacia	PT	NA
16	Fe, 24	15	A: electrification on head, numbness of left hand, feeling of landslide, walking on space,bilateral hand pain.	SPS SGTCS	R TO paroxysmal discharges	R posterior P cystic lesion	Encephalo-malacia	MT	Seizures 2/y in 3 years of follow-up
17	Fe, 18	13	Encephalitis at 9 months-old, Mental-motor retardation A: Yawing, crying, fear expression S: bilateral limb convulsions	CPS SGTCS	1. Generalised background anomaly 2, 3. N, N 4.Bifrontal epileptiform anomaly Bilateral P hyperintensi-ties, encepha-litissequela	Sequela encephalitis	PT	NA	
18	Fe, 21	7	head injury at 11 months-old A: - S: left focal onset secondary generalised seizures (vocalisation during sleep than seizure)	SGTCS	1,2 R P slowing of background rhythms 3.T dysryhthmia during HPV 4,6. N 5.Anterior paroxysmal discharges duringHPV 7,8.CT paroxysmal 9.CT epileptiform paroxysmal discharges	R P cystic Encephalo-malacia	Stroke	MT	Seizure free for 9 years
19	Fe, 65	60	A: Right sided headache, vertigo, anosmia, hypogeusia	SPS	R T background rhythm anomaly	R Post P subacute infarct	Stroke	MT	Exitus cardiopul-monary arrest
20	M, 71	1	2 mo meningitis A: R arm pain S: R arm clonic convulsion	SPS SGTCS	L CT paroxysmal anomaly at HPV	L P chronic infarct	Stroke	PT	NA
21	M, 19	4	Menengitis at 1 yo A: vertigo, fatigue S: turning around himself for 2-4 tours, oral automatism, gulping, forced deviation of head to the left then falling	SPS CPS	R FT epileptiform abnormality	R P infarct	Stroke	PT	NA
22	Fe, 48	4	A: - Fainting when face with blood, GTCS (eyes are open, pallor, head deviation to the back for 5-6 seconds)	CPS Reflex epilepsy	1.Paroxysmal anomaly with HPV 2.-4. N,N,N 5. Paroxysmal anomaly at vertex and temporal regions	L P chronic infarct	Stroke	MT	Seizures continue in 7 years of follow-up
23	Fe, 60	60	A: numbness of peroral region and hand	SPS	N (2 times)	P subcortical cystic infarct	Stroke	MT	Seizure free(6 y)

Table 1. Characteristics of 46 lesional symptomatic parietal lobe epilepsy patients (continued)

No	Sex/ age on admission	Onset	Medical History	Seizure type	Surface EEG	CT and /or MRI	Diagnosis	Treatment (surgery, RT, ChT, AED)	Prognosis
24	M, 30	30	Head injury at 4 yo A: Epigastric raising, vertigo, faint, nause, sour taste, falling	CPS SGTCS	R T paroxysmal anomaly	P leptomeningeal cyst, Posterior P chronic infarct	Stroke	MT	NA
25	M, 24	14	4 yo head injury A: epigastric sensation, gastric pain, headache and loss of consciousness (gastric, gall bladder operations because of pain)	SPS SGTCS	1.R T paroxysmal epileptiform anomaly 2. R TO epileptiform anomaly 3.R TO epileptiform anomaly 4-10. N	R P cortical-subcortical infarct	Stroke	PT	Seizures 1/year under AED treatment
26	M, 32	17	A: Feeling cold, confusion, oral-hand automatism GTCS on sleep	CPS SGTCS	L TP paroxysmal anomaly	R P subdural gyrus hyper-intensities	Stroke	MT	NA
27	Fe, 67	61	A: fainting Left hemiparesis	CPS GTCS	1.N, 2.T paroxysmal anomaly 3.L T slow waves, background anomaly	L P chronic ischemic lesions	Stroke (amiloidangiopathy)	MT	Exitus 10 y after 1st seizure (respiratory arrest)
28	Fe, 14	3,5	Head injury at 6 mo A: Vertigo, macropsy, left arm numbness S: Tonic posture of left body, vocalisation	SPS CPS	1.R PO background anomaly, epileptiform anomaly 2.R PO paroxysmal anomaly	R P dysplasia	MCD	MT	SPS 1/month
29	M, 49	44	A: Fatigue, blurred vision, vomiting S: Loss of consciousness	SGTCS	N	L posterior P gyrus thickness	MCD	MT	Seizure free under treatment
30	M, 17	17	A: numbness of left arm, S:SGTCS on sleep	SPS SGTCS	Posterior paroxysmal anomaly	R P closed schisencephaly	MCD	PT	NA
31	M, 22	2	A: Febril convulsion Clonic convulsion on right legGTCS on sleep, vocalisation	SPS GTCS	1. Background abnormality, C and L paroxysmal anomaly 2.L CT paroxysmal anomaly, 3,4. N	L P closed schisencephaly, polymicrogyri	MCD	PT	NA
32	Fe, 18	1	Infantil spasm at 20 day-old A: psychic+vizüel (fear, palpitation, black foals on visual field) S: Contraction on left eyelid, left arm, falling, seizures on sleep	SPS CPS SGTCS	1. R C paroxysmal abnormality 2. R FC epileptiform abnormality 3. R CTP epileptiform abnormality	R P polymicrogyri	MCD Pathology Cortical dysplasia	Lesionectomy PT	Seizures 1/month
33	M, 21	19	A: Epigastric sensation S: automatism, SGTCS	CPS SGTCS	1. R CT epileptiform abnormality 2. R T epileptiform abnormality	R P cortical dysplasia	MCD Pathology Cortical dysplasia	Lesionectomy PT	NA
34	M, 16	13	Café au lait on R leg A: Tongue numbness, bilateral hand numbness, blinking	SPS CPS	1. C, 2. L FT epilep-tiform abnormality 3.L FC paroxysmal anomaly	1. N (1999) 2. L P fold asymmetry (2001)	MCD	MT	SPS/ 6 months
35	Fe, 17	5	A: Nause, headache S: Black heterogenous flag on vision field, vomiting, falls at 15 yo, myoclonic jerks on legs, shivering, atonic seizures. Oral automatism, myoclonic jerks during sleep.	SPS CPS SGTCS	Bilateral O epileptiform abnormality	Biparietal atrophy	Atrophy	MT	NA

Table 1. Characteristics of 46 lesional symptomatic parietal lobe epilepsy patients (continued)

No	Sex/ age on admission	Onset	Medical History	Seizure type	Surface EEG	CT and /or MRI	Diagnosis	Treatment (surgery, RT, ChT, AED)	Prognosis
36	M, 30	25	Head injury at 2 yo A: - S: Bilateral convulsions on arms lasting 15 s	CPS SGTCS	Bilateral F paroxysmal anomaly	Biparietal atrophy (R> L)	Atrophy	MT	NA
37	M, 21	7	A: confusion, feeling empty, can not find his way, blinking S: Left face, arm and leg contraction on sleep	SPS SGTCS	R C epileptiform anomaly	R P focal cortical atrophy	Atrophy	PT	NA
38	M, 18	1	Head injury at 1 mo A: Numbness of left side, round shape lights coming from left vision field S: Staring, tonic posture of left hand than convulsion	SPS CPS SGTCS	Bilateral background abnormality	R P atrophy	Atrophy	PT	NA
39	M, 26	26	A: psychic symptoms (annoyance, feeling dead, strange thoughts, panic, unable to breathe) S:Bilateral clonic act of legs	SPS CPS	1.L T diffuse slow wave activity 2. R>L FCT epileptiform activity on sleep deprivation	Biparietal Atrophy	Atrophy	MT	NA
40	M, 38	36	A: Nausea, numbness of left arm S: Clonic convulsion of left arm. SGTCS on sleep.	SPS SGTCS	1.R T background abnormality, epileptiform abnormality	R P AVM	AVM	AVM embolization MT	SPS /4 month
41	Fe, 25	25	Menengitis at 13 yo A: Severe headache, numbness of left body side, anxiety, vertigo S: Right arm clonus, unable to talk	SPS SGTCS	1,2. L CTP background anomaly, epileptiform abnormality with HPV 3.L TPO active focal epileptiform anomaly	L P AVM	AVM	PT	NA
42	Fe, 24	23	Febril convulsion at 3 yo A: left face, arm numbness, blurred vision S: right arm numbness, right leg clonic convulsion SGTCS	SPS SGTCS	1,4. L CT background abnormality 2,5. L O, L FCT epilepti-iform abnormality 3. Subcortical epileptiform abnormality	L Posterior P AVM	AVM AVM embolization performed	PT	NA
43	M, 33	30	A: oral bad smell, feeling neck compression then GTCS	SPS SGTCS	1. N	L P caver-noma	AVM	MT	NA
44	Fe, 24	24	A: nausea, right arm numbness S: right arm tonic convulsion on sleep then SGTCS	SPS SGTCS	1. N	L P cavernous angioma	AVM	PT	NA
45	Fe, 24	20	A: Feeling empty S: Block of speech, staring, loss of contact, oral-hand automatism	CPS	L>R T paroxysmal abnormality	L P cavernous angioma	AVM	PT	NA
46	M, 28	21	A: - GTCS on sleep	GTCS	N	L P vascular abnormality	AVM	MT	NA

Note: Patients without aura were colored in grey. A: aura; AED: antiepileptic drug; AVM: arteriovenous malformation; C: central; ChT: chemotherapy; CPS: complex partial seizure; CT: computerized tomography; F: frontal; Fe: female; M: male; HPV: hyperventilation; L: feft; MCD: Malformation of Cortical Development; MRI: magnetic resonance imaging; MT: monotherapy; N: normal; NA: not available; O: occipital; P: parietal; PLED: Periodic Lateralised Epileptiform Discharges; PT: polytherapy; R: right; RT: radiotherapy; S: seizure; SGTCS: secondary generalized tonic clonic seizure; SPS: simple partial seizure; T: temporal; yo: years old

The aim of this study was to identify the clinical, electrophysiological and neuroimaging characteristics of patients with medically refractory symptomatic parietal lobe epilepsy. These findings may help us to localize the seizure focus in some patients with cryptogenic partial epilepsy and warn clinicians when the parietal lobe should be sampled with more invasive techniques like intracranial electrodes prior to epilepsy surgery.

METHODS

We performed a retrospective, descriptive study of patients who underwent a comprehensive evaluation including clinical, electroencephalographic and neuroimaging procedure for intractable lesional parietal lobe epilepsy for 11 years at the Department of Neurology of our hospital. This study

was conducted in accordance with Helsinki Declaration. All patients had parietal lesions and most of them were not candidate for epilepsy surgery for example tumor or arteriovenous malformation patients. That is why patients were not chosen from long-term video-EEG monitoring pool which is basically used for epilepsy surgery patients. Their detailed clinical history, neurological examination, routine surface EEG and neuroimaging features were noted. If patients' seizure semiology and brain lesions were indicative of parietal lobe epilepsy, they were included in this study. The parietal lobes have arbitrary anatomical borders. So we used the term "parietal lobe" to refer to the region behind the post central gyrus and in front of the occipital lobe according to the neuroimaging data (5). Patients were excluded if the lesion was large and extended beyond the parietal lobe and patients with dual pathology like an arachnoid's cyst or venous anomaly, which could be coincidentally found, were also excluded.

Patients' past medical history including febrile convulsions, family history of epilepsy and parental consanguinity were noted. All patients underwent a routine interictal scalp EEG, at least once, using the international 10-20 system. EEG recordings were interpreted by experienced epileptologists-neurophysiologists.

Nineteen patients had both brain computerized tomography (CT) and magnetic resonance imaging (MRI), 7 patients had only brain CT, the rest of them (23 patients) had only brain MRI scanning (including T1-weighted, T2-weighted, and fluid-attenuated inversion recovery [FLAIR] sequences).

Patients' follow up were performed by either routine control examinations or by phone call.

Statistical Analysis

SPSS 16.0 (Statistical Package for the Social Sciences Inc. released 2007. SPSS for Windows, version 16.0, Chicago, USA) was used for statistical analysis. Age data were presented as median and minimum-maximum levels. The patients' gender, medical history, types of aura, seizure types, ictal characteristics, presumed etiological factors and lesion side were shown as frequencies and percentages. Patients without aura and presumed etiological factors were compared by Kruskal-Wallis test. Mann-Whitney U test was used when appropriate. A value of $p<0.05$ was considered statistically significant.

RESULTS

Patients admitted to our clinic with medically refractory seizures and diagnosed as lesional parietal lobe epilepsy were included in this descriptive study. We combined their medical records and seizure semiology (aura and ictal characteristics) with EEG and neuroimaging results to obtain a clue guiding us to parietal foci.

At the time of data collection, there were 46 patients (21 female, 25 male) aged 14 to 71 years (median 24) who had lesional parietal lobe epilepsy. Their age at seizure onset ranged from 1 to 61 years (median 18). Their medical history, seizure semiology, surface EEG and neuroimaging characteristics are summarized in Table 1.

According to their past medical history, the rate of febrile convulsions was 21.7% (10/46), family history of epilepsy was 23.9% (11/46) and parental consanguinity was 8.7% (4/46) (Table 2.1).

Auras

Auras were reported in 78.3% (36/46) of the patients. The most common one was somatosensory aura described by 72.2% (26/36) as tingling or numbness; they were contralateral to the lesion side in 73.0% (19/26) patients, and bilateral in 2 patients. Three patients described pain including left facial pain, leg pain, bilateral hand and gastric pain. Interestingly, the patient with gastric pain had gastric and gall bladder operations because of this symptom before the diagnosis of epilepsy. Two patients had a cold sensation contralateral to the lesion side. Other sensory auras were related to taste, such as sour-tasting or smell including anosmia or bad odor.

The second most common aura was headache reported by 36.1% (13/36) of the patients. The description of headache was like wearing a helmet or headache without a specific feature.

Nausea and vomiting were detected in 36.1% (13/36) of patients and again 36.1% (13/36) of the patients reported psychic symptoms including fear expression, feeling empty, annoyance, feeling dead, strange thoughts, panic, anxiety and de ja vu.

Visual symptoms were described by 16.6% (6/36) of the patients, as mottled flag, round shaped lights, black foals on visual field, blurred vision, flashing and macropsy. Vertigo was reported as an aura in 11.1% (4/36) of the patients. One patient mentioned dysarthria as an aura symptom (Table 2.2).

Case Presentations

Patient 1: A 14-year-old female patient was admitted to our neurology clinic for having seizures. Her past medical history revealed that she had head injury at 18 months of age and her first seizures started at the age 3.5. She had aura with vertigo, numbness in the left arm and feeling of her eye bigger than before. Her seizure characteristics were tonic posture of left arm and leg, vocalization without affecting consciousness lasting 15-20 seconds. EEG showed right parieto-occipital background abnormality and epileptiform abnormality. Her cranial CT was normal at that time. Her seizures were controlled with carbamazepine (CBZ) and diphenylhydantoin (DPH) for 6 years, then antiepileptic medication was discontinued. After that, seizures started with a frequency of 20 times a day. EEG revealed right parieto-occipital background abnormality and centro-temporal paroxysmal activity. Her cranial MRI was normal. Then she was put on CBZ and primidone (PRM) treatment. After 17 years of her seizure onset, her thin section cranial MRI revealed right parietal cortical dysplasia. She still has seizures starting with left arm numbness and weakness, then tonic posture once a month under antiepileptic treatment.

Patient 2: A 24-year-old, male patient was admitted to our clinic for headache and seizures. He had a history of cranial trauma at the age of 4, which caused unconsciousness. His seizures started when he was 14 years old. Seizure characteristics were gastric pain, epigastric raising, headache then secondary generalized tonic clonic seizures (GTCS). He also had seizures in sleep. During follow up he had gastric and gall bladder operations because of abdominal pain, which in fact was a seizure aura. His EEGs revealed right temporo-occipital epileptiform abnormality. Cranial CT showed right parietal infarct and there was right parietal cortical-subcortical infarct sequela in MRI. Carbamazepine and DPH treatment were given to the patient and his seizure frequency decreased to 2 times/year.

Objective Seizure Manifestations and Seizure Types

The most common ictal behavioral changes were paresthesia in 69.6% (32/46) and focal clonic activity was present in 39.1% (18/46) of the pa-

Table 2. Patient characteristics, aura features, ictal symptoms, seizure types, interictal EEG findings and presumed etiological factors

1. Medical history of patients		**4. Seizure types**	
Febrile Convulsions	21.7%	Simple partial seizures	69.6%
Family History of Epilepsy	23.9%	Secondary generalised tonic clonic seizures	58.7%
Parental Consanguinity	8.7%	Complex partial seizures	43.5%
2. Aura		**5. Interictal EEG findings**	
Sensory symptoms	72.2%	Lateralised paroxysmal activity	56.5%
Headache	36.1%	Epileptiform abnormalities	34.8%
Nausea vomiting	36.1%	Abnormal background activity	33.3%
Psychic symptoms	36.1%	Normal EEG	34.8%
Visual symptoms	16.6%		
Vertigo	11.1%		
Others (smell, taste, pain, thermal, dysarthria)	26%		
3. Ictal behavioral changes		**6. Presumed etiological factors**	
Paresthesia	69.6%	Posttraumatic encephalomalacia	21.7%
Focal clonic activity	39.1%	Stroke	21.7%
Tonic posture	17.3%	Tumor	15.2%
Automatisms	17.3%	Malformation of cortical development	15.2%
Staring	11.1%	Arteriovenous malformation	15.2%
Speech disturbances	8.7%	Atrophy	10.9%
Vocalisation	8.7%		
Others (Head deviation, sensation of pain)	15%		

tients (Table 2.3). Tonic posture, various automatisms, head deviation, staring, sensation of pain and speech disturbances occurred to a lesser extent.

Simple partial seizures were the most common seizure type presenting in 69.6% (32/46) of the patients. Complex partial seizures occurred in 43.5% (20/46) and secondary GTCS were reported in 58.7% (27/46) of the patients (Table 2.4).

EEG Findings

Interictal EEG disclosed abnormal background activity in almost 1/3 of the patients (Table 2.5). Epileptiform abnormalities were found in 34.8% (16/46) and lateralized paroxysmal slow, sharp-slow waves were detected in 56.5% (26/46) of the patients. EEG findings were normal in 34.8% (16/46) of the patients. In none of the EEGs isolated parietal foci were detected.

Presumed Etiological Factors

Posttraumatic encephalomalacia 21.7% (10/46), stroke 21.7% (10/46), tumoral lesion 15.2% (7/46), malformation of cortical development 15.2% (7/46), arteriovenous malformation 15.2% (7/46) and atrophy 10.9% (5/46) were detected in decreasing order (Table 2.6). Lesion localizations were as follows: right hemisphere in 47%, left hemisphere in 44% and bilateral parietal atrophy in 9% of the patients.

Is the Presence of Aura Related to the Etiology?

In this study, 10 patients (21.7%) did not present aura symptoms before seizure manifestation. Most of the patients without aura had tumoral lesions (5/10 patients). This finding was statistically significant (p=0.02). In the rest of them, two patients had stroke, one had malformation of cortical development, one had parietal atrophy and another had arteriovenous malformation. Age, seizure types, EEG characteristics, side of the lesion and antiepileptic treatment were not different between patients with or without aura (p>0.05).

Surgery

Seven of 46 patients had tumoral parietal lobe lesions and 6 of them underwent surgical procedures. Pathological examination showed that 4 of 6 patients had astrocytoma, one had low-grade glial tumor and one had meningioma. The seventh patient had nodular lesion with contrast enhancement. The pathological diagnosis of these 6 patients is shown in Table 1. After surgery 3 of them received chemotherapy with radiotherapy, one was treated with radiotherapy only. They were also treated with two or three antiepileptic drugs for their seizures. The patient with meningioma became seizure free under antiepileptic medication (DPH+CBZ) followed by tumor resection. Postoperative follow-up duration was one year.

Seven of 46 patients had malformation of cortical development and lesionectomy was performed in two of them. Pathological investigation revealed focal cortical dysplasia. After surgery one of them had one seizure per month under multiple antiepileptic drug treatments. In the remaining 5 patients who did not have surgery, one was seizure free, one had a simple partial seizure (SPS) per month and one had a SPS per 6 months while taking one antiepileptic drug (AED).

One patient in the arteriovenous malformation group underwent embolization. Unfortunately, her follow-up was not available. Totally 19.5% (9/46) of patients had surgical interventions because of their parietal lesions.

Prognosis

Twenty patients' follow-up records were obtained. Follow-up time ranged between 1-10 years. Five patients were seizure free under AED treatment. Thirteen patients were having seizures either simple partial or secondary GTCS while taking AED treatment. Two patients died because of cardiopulmonary arrest and respiratory failure during 10 years of follow-up. Detailed information is provided in Table 1.

DISCUSSION

Literature about parietal lobe seizures is limited to case reports or case series. Especially reports related to aura and seizure semiology with parietal localization are rare (3,5,8,11). Here we presented the clinical and laboratory findings of 46 lesional symptomatic parietal lobe epilepsy patients. In this retrospective study, the main characteristic of parietal lobe seizures was the aura compatible with literature. The most common auras encountered were somatosensory; and 72.2% of patients experienced tingling or numbness of the extremities. Somatosensory auras have been assumed as the most common initial manifestations in patients with symptomatic parietal lobe epilepsy in the literature (4,5,12). Somatosensory aura was contralateral to the epileptogenic side in 73%, painful sensations were described in three patients and thermal sensations in two. Several authors have reported the lateralizing value of somatosensory aura in the literature (13). Painful sensations may also represent as an epileptic aura. In 1 of 3 patients the aura was contralateral to the epileptic side and in 2 of them painful sensations were bilateral. These signs may reflect the involvement of the secondary sensory areas. Although these sensory symptoms give an impression that the patient may have a parietal lesion, sometimes parietal lobe seizure manifestations are various and complex. The parietal lobe is the interface of all tracts coming from or going to other brain regions, especially to the frontal and temporal lobes (3,14). That is why parietal lobe seizures may have different kind of features like visual, gustatory, motor, psychic etc. It was the case also in our study. Vestibular manifestations were reported in 11-23.5% of the series presented in the literature (5,8). They probably reflect the involvement of the vestibular cortex (15). Visual aura may show the spreading nature of seizures to the occipital lobe. In general, subjective manifestations of aura symptoms were present in 78.3% of the patients in our study. The rate of subjective manifestations in parietal lobe epilepsy ranges between 57% to 94% in the literature (3,5,7,11), which is in agreement with our findings.

In this retrospective study, the most common ictal behavioral changes were paresthesia and focal clonic activity. Tonic posture, various automatisms, head deviation, staring, sensation of pain and speech disturbances occurred to a lesser extent. These results again were in accordance with the literature (5,8,16).

Among localization related epilepsies, parietal lobe seizures have the highest percentage of non-localizing EEG findings, probably owing to the rich connectivity of the parietal lobe (3,9). In our study we also observed the same finding. None of our patients' EEGs revealed isolated parietal foci. This may explain the mislocalization of non-lesional parietal lobe epilepsy to other lobes in some cases.

According to our study, the most common presumed etiological factors were posttraumatic encephalomalacia and stroke making up nearly half of the patients. The rest of them were tumor, malformation of cortical development, atrophy, arteriovenous malformation. These results were compatible with previous studies (3,5,8). In this study we found that most patients without aura had tumoral lesions. This finding contradicts with the literature, where aura is reported in up to 90% of the patients with tumoral parietal lobe epilepsy. The reason for this discrepancy may be the low number of tumoral patients in our study.

Roughly 30% of epilepsy patients have medically refractory seizures (17). For some treatment resistant focal epilepsy patients, surgical excision of the epileptic focus is an alternative choice to medical treatment (5). Although the parietal cortex comprises the second-largest cortical surface of the brain and despite the significant progress in video-EEG recording technology, imaging techniques, and invasive mapping methods with depth and/or subdural grid electrodes, resections in the parietal lobe are rare, constituting only 10.8% of the patients in large surgical series (5,18). In our study 19.5 % of patients had surgical intervention which is comparable to the literature.

This study emphasizes the importance of the symptomatology of the parietal lobe seizures. However, it has some limitations. One drawback of the study is the presentation of only routine scalp EEG findings. Besides patients were not chosen from long-term video EEG monitoring or intracranial electrode implant pool, so that parietal foci were verified only with neuroimaging data. Also follow-up of all patients was not available. Despite these limitations, 46 patients of lesional parietal lobe epilepsy patients' information is valuable for the epilepsy literature.

In conclusion, clinical and laboratory findings of patients with parietal lobe epilepsy are scarce in the literature. This relatively large study results indicate that parietal lobe seizures may have different symptomatology, owing to various patterns of seizure spread. Interictal scalp EEG recordings contain non-localizing epileptiform abnormalities in only 1/3 of the patients. Sensory aura is the most predictable sign of parietal lobe origin. Medically refractory cryptogenic partial epilepsy patients, who have various sensory symptoms like lateralized paresthesias or pain, should be investigated for parietal seizure foci.

Ethics Committee Approval: Authors declared that the research was conducted according to the principles of the World Medical Association Declaration of Helsinki "Ethical Principles for Medical Research Involving Human Subjects", (amended in October 2013).

Informed Consent: As this study was a retrospective one and medical file records were scanned, informed consent was not obtained.

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